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The relative contribution of nmdars to excitatory postsynaptic currents is controlled by ca2+-induced inactivation

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Abstract

© 2016 Valiullina, Zakharova, Mukhtarov, Draguhn, Burnashev and Rozov. NMDA receptors (NMDARs) are important mediators of excitatory synaptic transmission and plasticity. A hallmark of these channels is their high permeability to Ca2+. At the same time, they are themselves inhibited by the elevation of intracellular Ca2+ concentration. It is unclear however, whether the Ca2+ entry associated with single NMDAR mediated synaptic events is sufficient to self-inhibit their activation. Such auto-regulation would have important effects on the dynamics of synaptic excitation in several central neuronal networks. Therefore, we studied NMDAR-mediated synaptic currents in mouse hippocampal CA1 pyramidal neurons. Postsynaptic responses to subthreshold Schaffer collateral stimulation depended strongly on the absence or presence of intracellular Ca2+ buffers. Loading of pyramidal cells with exogenous Ca2+ buffers increased the amplitude and decay time of NMDAR mediated EPSCs (EPSPs) and prolonged the time window for action potential (AP) generation. Our data indicate that the Ca2+ influx mediated by unitary synaptic events is sufficient to produce detectable self-inhibition of NMDARs even at a physiological Mg2+ concentration. Therefore, the contribution of NMDARs to synaptic excitation is strongly controlled by both previous synaptic activity as well as by the Ca2+ buffer capacity of postsynaptic neurons.

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Keywords

Action potentials, Calcium, Excitation, Firing properties, Modulation, NMDA receptor